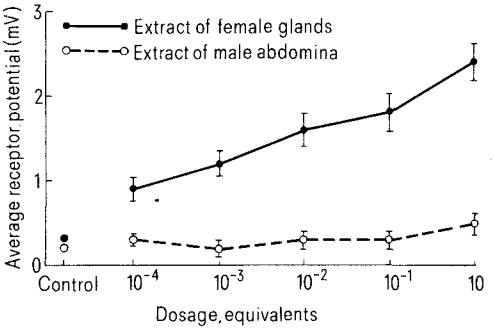

Figure 2. Receptor potentials recorded from olfactory sensilla on the terminal antennal segment of male *Lasioderma serricorne* F. evoked by surface extracts (hexane) of intact female glands and male abdomina.
Stimulation was carried out by passing air puffs (20 ml/0.5 sec) over graded levels of the above extracts. Extracellular DC potentials were recorded by glass electrodes (microcapillaries) containing Beadle-Ephrussi's Ringer solution. Each point on the curves represents the average receptor potential recorded from eight antennae and the relevant standard deviation (vertical line).

equivalents of a hexane extract of abdomina excised from unmated male *L. serricorne* caused receptor potentials of 0.3 and 0.5 mV respectively, which failed to differ significantly from the control values (stimulation by air puffs).

Premating behavior. Bioassays[11] on circular filter papers (diameter 75 mm) impregnated in the central region (diameter 3 mm) by a hexane extract of intact glands displayed the attractiveness of the latter for unmated male tobacco beetles. The tests involved responses of 100 insects exposed in groups of 10 to the above experimental conditions and revealed the following average results: 1 gland equivalent of the above extract induced 39.7 (s.d. = 1.8) visits of male *L. serricorne* on the impregnated region, while 10 gland equivalents increased this parameter to 51.2 (s.d. = 2.4) and also caused several attempts of copulation, within an observation period of 15 min. However, a surface extract (hexane) of virgin females proved to be significantly more attractive than the gland extract inducing 62 (s.d. = 2.7) visits and repeated attempts of copulation, when compared at the level of one equivalent. This is probably due to repeated pheromone secretion from the gland resulting in pheromone accumulation in the cuticle lipids of virgin females. Moreover, the mean receptor potentials induced by the surface extract of virgin females were significantly higher than those elicited by the gland extract: one equivalent of the former induced 3.2 ± 0.3 mV, whereas one equivalent of the latter elicited 2.4 ± 0.2 mV in the antennae of male tobacco beetles.

Interestingly, the behavior of male tobacco beetles induced by a hexane extract of homogenized glands was found to differ from that observed in presence of a hexane extract of intact glands: although unmated males rapidly approached the region impregnated with extract of the homogenate (1 or 10 gland equivalents), they left it instantaneously followed by a sharp turning. It appears that the extract of intact glands merely provides the sex pheromone emitted from the orifice (fig. 1a), whereas the extract of homogenized glands also comprises less volatile components and/or metabolites evoking the repulsion of male tobacco beetles.

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1 Chuman, T., Kohno, M., Kato, K., and Noguchi, M., Tetrahedron Lett. *25* (1979) 2361.
2 Chuman, T., Kato, K., and Noguchi, M., Agric. biol. Chem. *43* (1979) 2005.
3 Ono, M., Onishi, I., Chuman, T., Kohno, M., and Kato, K., Agric. biol. Chem. *44* (1980) 2259.
4 Mori, K., and Nomi, H., Tetrahedron Lett. *22* (1981) 1127.
5 Mori, K., Chuman, T., Kohno, M., Kato, K., and Noguchi, M., Tetrahedron Lett. *23* (1982) 667.
6 Mori, M., Chuman, T., Kato, K., and Mori, K., Tetrahedron Lett. *23* (1982) 4593.
7 Chuman, T., Kohno, M., Kato, K., Noguchi, M., Nomi, H., and Mori, K., Agric. biol. Chem. *45*, (1981) 2019.
8 Hoffman, R.W., Helbig, W., and Ladner, W., Tetrahedron Lett. *23* (1982) 3479.
9 Coffelt, J.A., and Burkholder, W.E., Ann. ent. Soc. Am. *65* (1972) 447.
10 Levinson, H.Z., Levinson, A.R., Francke, W., Mackenroth, W., and Heemann, V., Naturwissenschaften *68* (1981) 148.
11 Levinson, H.Z., Levinson, A.R., Francke, W., Mackenroth, W., and Heemann, V., Naturwissenschaften *69* (1982) 454.
12 Chuman, T., Mochizuki, K., Mori, M., Kohno, M., and Kato, K., Agric. biol. Chem. *46* (1982) 593.
13 Chuman, T., Mochizuki, K., Mori, M., Kohno, M., Kato, K., Nomi, H., and Mori, K., Agric. biol. Chem. *46* (1982) 3109.
14 Romeis, B., Mikroskopische Technik, 16th edn. Oldenbourg, München/Wien 1968.
15 Levinson, A.R., Levinson, H.Z., Schwaiger, H., Cassidy, R.F. Jr, and Silverstein, R.M., J. Chem. Ecol. *4* (1978) 95.
16 Dailey, R.J., and Happ, G.M., J. Morph. *171* (1982) 259.

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Fat-supplemented diet protects against activity-stress ulcers in rats[1]

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Summary. Rats were exposed to a fat-supplemented (32.5% beef tallow) diet from weaning until they were 200 g in weight. Half the animals were exposed to the activity stress paradigm consisting of housing in standard activity-wheel cages while restricting their food intake to 1 h each day. Control rats were treated in the same fashion but on a fat-free (0.08%) diet. Fat-supplemented activity-stressed rats exhibited high levels of running wheel activity but less mortality and less gastric pathology than fat-free controls which were exposed to the activity-stress procedure. Ulcers were not observed in home cage housed rats in either diet condition.

If young adult male rats are housed in standard laboratory activity wheel cages and fed only 1 h each day, they will die within 7–10 days and reveal massive gastric glandular ulcers[2–4]. Much of this gastric damage is indeed true ulcer, penetrating the muscularis[5]. Home cage housed rats which are also fed only 1 h per day (food 'yoked' to the wheel-

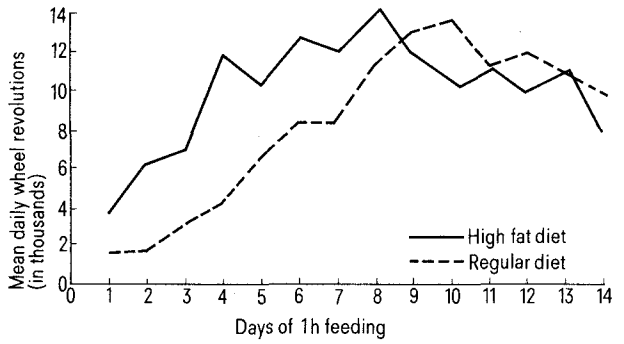

Figure 1.

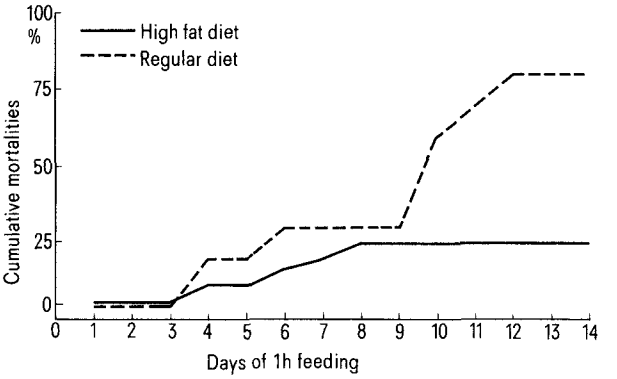

Figure 2.

housed animals and given exactly the same amount of food to eat as the wheel housed rats ate in 1 h) do not die and do not exhibit any signs of gastric disease. Because activity is an important factor in this procedure, it has been called the 'activity-stress' ulcer model. The cause of death of activity-stress animals is unclear. However, clinical evaluation of these rats revealed a gradual decline in core body temperature[6], decreased liver glycogen, decreased blood glucose, cholesterol and total protein as well as increased B.U.N.[7], indicative of hypoglycemia. Clearly, animals maintained on a restricted feeding regimen (1 h per day access to food) but which also engage in high levels of running wheel activity do not have sufficient caloric intake to maintain this behavior for long periods of time. One component of diet, fat, has been implicated in gastric disease. Raghavan[8] noted that of 31 cases of human duodenal ulcer, 19 cases consumed less that two-thirds of the recommended daily allowance of dietary fat.

Accordingly, the present study sought to examine the effects of a fat-supplemented diet on activity-stress-induced gastric disease in rats.

Method. 60 male Wistar rats born at the University of Winnipeg animal colony were used. They were first randomly divided into a fat-supplemented diet group and a low fat control group. When the animals reached 21 days of age, they were weaned, housed in individual cages and fed either a fat-supplemented diet (addition of 32.5% beef tallow, Bio-Serv Inc., Frenchtown, New Jersey, USA) or control diet (0.08% fat). When the animals reached 200 ($\pm$ 10) g, the activity-stress phase of the study began. Each of the fat-supplemented and control diet groups was further divided into 2 groups. 15 fat-supplemented diet animals and 15 low fat control rats were housed individually in standard activity-wheel cages (Wahmann Mfg. Co., Model LC-34) which also include an adjoining cage measuring $25 \times 15 \times 13$ cm. The remaining 15 animals in each of the 2 diet conditions served as controls and were kept in standard laboratory cages measuring $25 \times 17.5 \times 17.5$ cm. For 4 days, a habituation period occurred, during which time all rats had free access to their respective diets as well as to water. Beginning on the 5th day, and continuing for 14 days, all activity-stress rats were fed their respective diets for only 1 h per day (09.00–10.00 h). Water was always available ad libitum. The home cage rats in each diet condition were food-yoked to their wheel-housed counterpart. In this procedure, the exact amount of food eaten by a wheel-housed animal in the 1-h feeding period was fed to its home cage-housed partner during the next hour (10.00–11.00 h). In this manner, food intake remained constant between a wheel-housed rat and its home-cage-housed partner. When an activity-wheel animal died, its stomach was removed and examined for ulcers. Its food-yoked partner was killed at the same time and its stomach also examined. The location, number, and cumulative length (in mm) of any ulcers were recorded. In addition, trunk blood was collected and examined for glucose[9]. The study continued for 14 days at which time any surviving animals were killed and examined as described above.

Results. Figure 1 shows mean daily running-wheel activity in both the fat-supplemented and low fat groups. Running activity, a central component of the activity-stress ulcer model, was comparably high in both groups, and not significantly different between them. Figure 2 shows cumulative percent mortalities over the 14 days of activity stress. Low fat diet animals died more frequently and faster than did fat-supplemented animals. The table shows a summary of the stomach pathology and blood glucose data. Fat-supplemented wheel-housed rats displayed significantly fewer ($F(3,56) = 11.6$; $p < 0.01$; Tukey-test) and significantly less severe ($F(3,56) = 24.3$; $p < 0.001$; Tukey test) ulcers

Summary of stomach pathology* and blood sugar data for wheel-housed and control rats

Group	No. of rats	No. of rats with ulcers	No. of ulcers Mean ($\pm$ SE)	Cumulative ulcer length (mm) Mean ($\pm$ SE)	Blood glucose (mg/dl) Mean ($\pm$ SE)
High fat – wheel-housed	15	3	2.53 (0.39)	11.00 (1.91)	152.3 (4.70)
High fat – home cage-housed	15	0	0.00 (0.00)	0.00 (0.00)	124.9 (2.02)
Regular diet – wheel-housed	15	15	18.61 (7.66)	51.32 (8.17)	92.5 (5.02)
Regular diet – home cage-housed	15	0	0.00 (0.00)	0.00 (0.00)	139.6 (6.7)

*Glandular ulcers only.

than the low fat wheel-housed animals. No instance of gastric ulcer was observed in home cage animals in either diet condition. No evidence of hepatic lesions was observed in either control or wheel-housed animals. Blood glucose levels were significantly higher in the high-fat wheel-housed rats ($F(3,56)=5.60$; $p<0.01$; Tukey-test) and lowest in the regular diet wheel-housed animals. The temporal course of the blood glucose changes appeared to be that of a gradual decline over days of 1-h feeding. Rectal temperature (YSI Instruments) indicated that fat-supplemented activity-stress rats had slightly (but not significantly) higher core temperatures than low fat activity-stress animals (36.3 ± 0.9 °C and 34.3 ± 1.9 °C, respectively). Home cage control rats averaged 37.5 (±1.7) °C. The energy intake in the low fat groups (0.08%) was approximately 4.25 kcal/g of food per day, while that of the fat-supplemented group was approximately 7.35 kcal/g of food per day.

Discussion. These data reveal an important etiological factor in experimental activity-stress-induced gastric disease and suggest that dietary fat may have a prophylactic effect in the development of this disease. It is important to note that both groups of activity-stress animals (high fat and low fat diet groups) exhibited similar and elevated levels of activity. In fact, any observed activity differences between these groups were not statistically significant. Thus, the major difference between the groups was not one of activity, but rather one of diet. Supplementing the diet with 32.5% fat resulted in a marked reduction of ulcer incidence (3 out of 15 fat-supplemented diet rats showed ulcer disease as opposed to 15 out of 15 low fat diet animals), frequency, and severity. Whether this phenomenon is a local effect in the gut or a systemic effect (suggested by the blood glucose data) remains to be determined.

1 Supported by NSERC A8072. I thank K. Kiernan and C. Gustafson for technical assistance.
2 Paré, W., and Houser, V., Bull. Psychon. Soc. *2* (1973) 213.
3 Paré, W., Physiol. Behav. *16* (1976) 699.
4 Paré, W., Am. J. dig. Dis. *20* (1975) 262.
5 Manning, F., Wall, H., Montgomery, C., Simmons, C., and Sessions, R., Physiol. Behav. *21* (1978) 269.
6 Paré, W., Physiol. Behav. *18* (1977) 219.
7 Paré, W., Natelson, B., Vincent, G., and Isom, K., Physiol. Behav. *25* (1980) 417.
8 Skoryna, S., ed., Pathophysiology of Peptic Ulcer, p.465. McGill University Press, Montreal 1963.
9 Carroll, J., Smith, J., and Babson, A., Biochem. Med. *4* (1970) 171.

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Little-excitable transitional cells in the rabbit sinoatrial node: a statistical, morphological and electrophysiological study

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Summary. It is demonstrated that systolic and diastolic depolarization rate are correlated with the percentage of myofilaments in the cells of the rabbit sinoatrial node. It appears that, in the rabbit sinoatrial node, little-excitable transitional cells exist in the zone of propagation at the septal side of the typical nodal cells.

In a correlative electrophysiological and ultrastructural study of 42 cells in the rabbit sinoatrial node, we have found that this structure has a symmetrical morphology. From the ultrastructural point of view it comprises typical nodal cells, surrounded by transitional nodal cells which gradually become atrial cells at the periphery. However, its electrophysiological activation pattern is asymmetrical, showing a preferential conduction pathway of the impulse, diagonally upward to the crista terminalis[2]. This preferential pathway seems to be caused by the existence of a zone of little-excitable cells at the septal border of the sinoatrial node, rather than by poor electrical coupling[3]. It is clear from electrophysiological and electronmicroscopical observations that typical nodal cells have the following features: a low percentage of myofilaments, a low upstroke velocity of the action potential, and a prominent rate of diastolic depolarization. Atrial cells bordering the sinoatrial node have opposite features. The transitional cells between the atrial cells and the typical nodal cells are intermediate both in the percentage of myofilaments and in the rate of diastolic depolarization. It was observed that the rate of diastolic depolarization and the percentage of myofil-

Mean value of 6 variables (±SE) after distribution of 30 cells from the sinoatrial region over 4 classes of cell types by stepwise discriminant analysis

	Typical nodal cells	Normally excitable transitional cells	Little-excitable transitional cells	Atrial cells
Number of cells	9	10	10	1
Percentage of myofilaments	27 ± 1.5	34 ± 1.8	40 ± 2.7	41
Orientation of myofilaments (%)	21 ± 0.9	37 ± 2.0	54 ± 2.1	80
Organization of myofilaments (%)	37 ± 1.9	58 ± 1.4	68 ± 2.3	88
Rate of systolic depolarization (V/s)	10 ± 1.1	18 ± 1.5	12 ± 0.8	28
Rate of diastolic depolarization (mV/s)	50 ± 1.2	31 ± 0.7	14 ± 0.6	15
Maximal diastolic potential (mV)	−66 ± 1.6	−63 ± 2.6	−60 ± 4.4	−63

The percentage of myofilaments is the percentage of intracellular space occupied by myofilaments. The orientation and organization of the myofilaments are given as summed scores in percentages. Orientation: 100%, myofilaments in one direction only; 0%, myofilaments occurring in all directions. Organization: 100%, all myofilaments organized in myofibrils; 0%, all myofilaments occur isolated only.